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Case Report

Non-Arteritic Anterior Ischemic Optic Neuropathy Preceding Anterior Thalamic-Geniculate Perforator Infarct Induced Stroke: A Case Report

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Abstract

We report a sixty-one years old male, who presented with complaints of blurring of vision in his right eye for two weeks along with floaters and headache. Best corrected visual acuity (BCVA) was recorded as 20/30 and 20/20 in his right and left eye respectively. A relative afferent pupillary defect (RAPD) was noted in the right eye. Fundus examination revealed disc edema in the right eye with unremarkable fundus in the left eye. The color vision, contrast sensitivity, Amsler's grid evaluation and intraocular pressure (IOP) were within normal limits. Perimetry showed a superior altitudinal field defect in the right eye with unremarkable perimetry in the left eye. Keeping the diagnosis of non-arteritic anterior ischemic optic neuropathy (NA-AION), intravenous Methylprednisolone was started in pulse dose for three days. Appropriate investigations such as the Erythrocyte sedimentation rate (ESR) and complete blood count were ordered. After the completion of intravenous methyl-prednisolone for three days, the patient started complaining of weakness in the lower limbs and confusion. Neurological opinion was sought which revealed no significant abnormalities. The patient was sent to a cardiologist and an electrocardiogram (ECG) showed evidence of non-sustained ventricular tachycardia (NSVT). Magnetic resonance imaging (MRI) of the brain revealed an acute lacunar infarct involving the right thalamus (Right anterior thalamic-geniculate perforator territory infarct) with chronic ischemic microangiopathic changes. The patient was started on anti-hypertensive, anticoagulants, and oral steroids with the tapering dosage by the cardiologist.

Here is our aim to report a case of NA-AION which eventually led to a stroke that was induced by acute infarct of the anterior thalamic-geniculate perforator in an elderly patient.

Keywords: *Non-Arteritic Anterior Ischemic Optic Neuropathy; Disc Edema; Magnetic Resonance Imaging*

Introduction

Non-arteritic anterior ischemic optic neuropathy (NAION) is the most common cause of optic neuropathy above the age of 50 years [1]. The short posterior ciliary arteries passing through the scleral canal gets hypoperfusion which eventually leads to localized disc edema which further affects the collateral axons by leading them to ischemia and their functional loss by apoptosis [2]. Hypertension, hypercholesterolemia, diabetes mellitus, cardio- and cerebrovascular disease, and obstructive sleep apnea are very strong risk factors for NAION. The effect of systemic steroids on NAION is controversial but Hayreh., *et al*. reported the beneficial effect of systemic steroids in NAION by reducing the edema and inflammation of the optic nerve [3]. Here we are reporting this case as NAION can be the predictor for the silent stroke in elderly patients. If these cases can be detected well before time and treated properly can prevent eventual morbidities.

Case Presentation

A 61-year-old male patient presented to eye OPD with complaints of blurring of vision in his right eye for two weeks which was sudden in onset, non-progressive in nature, and constant throughout the day. It was associated with a right-sided headache for the same duration which was insidious in onset, non-progressive, dull aching in nature and relieved with sleep and medication. No known systemic history of migraine, hypertension, coronary artery disease, diabetes mellitus, tuberculosis, asthma, or epilepsy was present.

Examination revealed the best corrected visual acuity (BCVA) as 20/30 in the right eye and 20/20 in the left eye. There was a relative afferent pupillary defect (RAPD) noticed in the right eye. Fundus examination after pupillary dilatation revealed sectoral disc edema in the right eye. The left eye fundus examination was within normal limits with a cup disc ratio of 0.2 (Figure 1).

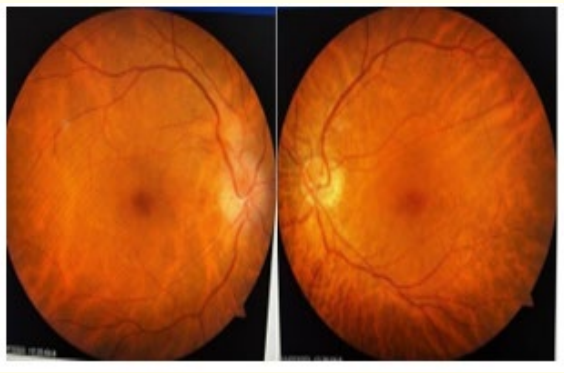

Figure 1: *Showing disc edema in the right eye and normal fundus of the left eye.*

Color Vision, Contrast Sensitivity, Amsler's grid evaluation and extraocular movements (EOM) were all within normal limits. The intraocular pressure (IOP) was 12 and 14 mmHg in the right and left eye respectively. Visual field charting (Humphrey field analyzer - 3) 30-2 showed a superior altitudinal field defect in the right eye and peripheral scotoma in the left eye (Figure 2). Intravenous Methylprednisolone I gm was started in pulse dose after getting unremarkable fasting blood sugar (FBS).

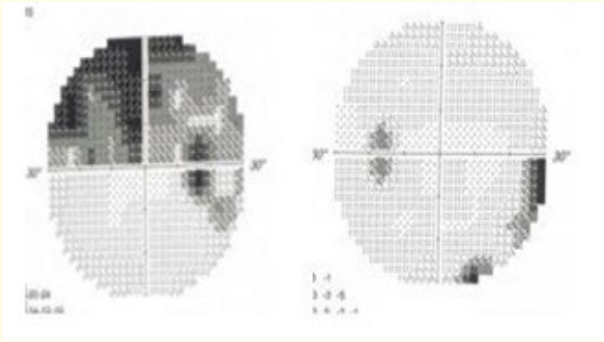

Figure 2: *Visual field of the right eye showing the superior altitudinal defect with the Peripheral scotoma of the left eye.*

OCT of the right optic nerve head (ONH) showed obscuration of the optic cup and elevated disc margins (Figure 3).

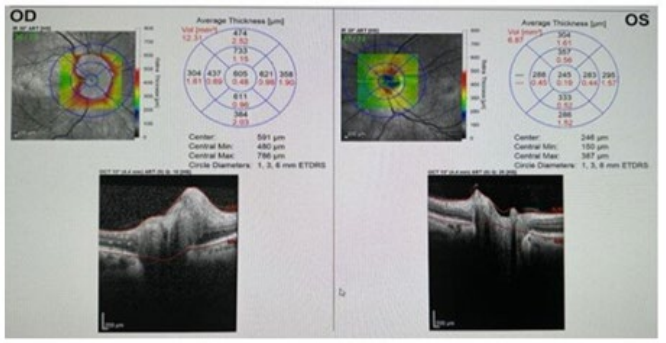

Figure 3: *Demonstrating disc edema in the right eye and normal optic nerve head on Optical Coherence tomography.*

VEP showed decreased P100 amplitude with normal latency.

His blood pressure was raised to 161/96 mmHg while rest vitals were within the normal range.

As the patient was a middle-aged man who presented with unilateral and painless diminution of vision, on examination was found to have disc edema and an altitudinal field defect. A provisional diagnosis of NA-AION was made and other appropriate investigations were ordered. The patient got symptomatic relief after the treatment. BCVA was 20/20 and the rest of the ocular examination was status quo. After one week patient reported back to us with weakness of his legs along with one episode of generalized body weakness. The patient was referred to the cardiology department for further evaluation. ECG was repeated for the patient which revealed borderline lateral T wave abnormality. Further Holter monitoring was advised which showed non-sustained ventricular tachycardia (NSVT).

MRI of the Brain was advised which showed an acute lacunar infarct involving right thalamus (Right anterior thalamo-geniculate perforator territory infarct) with chronic ischemic microangiopathic changes (Figure 4).

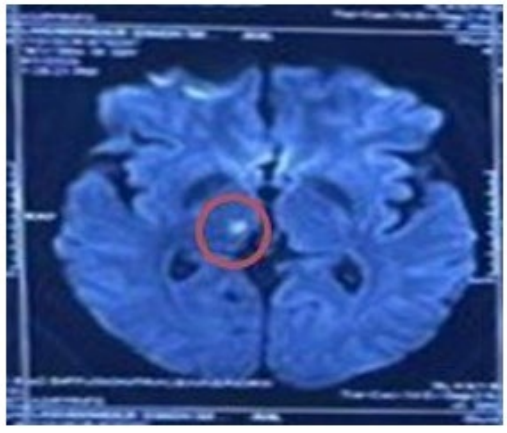

Figure 4: *Depicting the lacunar infarct in the right thalamus on magnetic resonance imaging.*

Non-contrast computed tomography (NCCT) Brain Angiography showed a lacunar infarct in the right thalamus with hypodense corona radiata suggestive of acute etiology.

Neurological examination revealed no significant findings.

The patient was started on anti-hypertensives, and anticoagulants and shifted to oral steroids with tapering dosage. Regular cardiac and neurological examinations closely monitored him.

On follow-up in the eye OPD, the patient was symptomatically better and the disc edema showed resolution.

Discussion

The presentation of posterior cerebral artery (PCA) strokes usually presents with a vast range of non-specific symptoms. Patients may be unaware of their symptoms or can even land in the emergency department. This increases the difficulty of establishing a diagnosis before any further damage can occur.

Anatomically PCA supplies blood to several major areas of the brain comprising the upper brainstem and midbrain, inferomedial temporal lobe (lower section of optic radiations), occipital lobe, and a large portion of the thalamus [4].

It is further divided structurally into 4 segments (P1-P4) such that the P2 segment includes thalamic-geniculate arteries (TGA) and the posterior choroidal arteries. The TGAs are responsible for the blood supply of the ventrolateral part of the thalamus. The posterior choroidal arteries supply the posterior thalamus, lateral geniculate body, pulvinar, hippocampus, and parahippocampal gyrus [4].

Very few studies have been done to determine that small vessel disease can lead to PCA infarcts. One of them is the Sagrat Cor Hospital of Barcelona Stroke Registry which concluded that lacunar infarcts were the most frequent subtype of stroke (34.5%) during a period of 19 years from 1986 to 2004 [5].

Various studies estimated that the incidence of PCA strokes is around 5% to 10%. Isolated PCA strokes were demonstrated to constitute 6.1% of cases of total strokes (n = 232/3808). The associated factors were also studied, for instance, mean age was found to be around 73.9 years and male preponderance was estimated to be 55.2% respectively [5,6]. These findings were consistent with the current case as the patient was male at the age of sixty-one years.

Small vessel arteriolosclerosis can occur due to aging or co-morbidities like hypertension and diabetes. The symptoms are based on the extent of occlusion and the location of the lesion ranging from mild blurring, headache, difficulty in focusing, diplopia, visual field changes (hemianopia, quadrantanopia, sectoranopia) to nausea, vomiting, aphasia, memory impairment, and behavioral disturbances that aide in localization of the lesion [6].

The lesion in the ventral posterolateral nucleus may result in pure sensory stroke which receives the blood supply from inferolateral TGA's.

The involvement of a single thalamic vascular territory was observed in 69% of patients in a study [7].

The study done by others demonstrated that the ventrolateral territory was the most frequently affected thalamic territory as seen in 61% of patients [4].

There was a significant association between involvement of the ventrolateral thalamus and isolated thalamic infarcts (48% (49/103) vs 28% (18/65); $p < 0.01$) [4,7].

A study conducted by Lee., *et al*. concluded that patients with NA-AION have increased chances of ischaemic stroke and more commonly with systemic co-morbidities (diabetes and hypertension) [8].

The visual acuity remains unchanged in the majority. Perforator artery strokes are difficult to diagnose as they have non-specific symptoms. The TGA branch of PCA (known as the ventrolateral PCA perforator) supplies the optic tract. Hypo-perfusion across ONH results in ischemic damage to ganglion cell axons [9].

PCA perforator artery strokes are difficult to diagnose due to non-specific symptoms. The TGA branch of PCA supplies the optic tract. Also known as the ventrolateral PCA perforator, it is most commonly affected in small vessel disease. Systemic workup of vasculopathy and control of risk factors is essential to prevent irreversible neurological sequelae. NA-AION results in ischemic damage to the axons of ganglion cells.

Conclusion

NA-AION can present as disc edema more commonly found in the elderly. It can predict a life-threatening thromboembolic phenomenon in a patient. PCA stroke is referred to as silent stroke. We can save a patient's life by timely diagnosis and cause-based treatment.

Declaration of Conflicting Interests

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Bibliography

1. Hayreh SS. "Ischemic optic neuropathy". *Progress in Retinal and Eye Research* 28.1 (2009): 34-62.
2. Archer EL, *et al*. "Obstructive sleep apnea and nonarteritic anterior ischemic optic neuropathy: evidence for an association". *Journal of Clinical Sleep Medicine* 9.6 (2013): 613-618.
3. Hayreh SS, *et al*. "Non-arteritic anterior ischemic optic neuropathy: role of systemic corticosteroid therapy". *Graefe's Archive for Clinical and Experimental Ophthalmology* 246.7 (2008): 1029-1046.
4. Lazzaro N A, *et al*. "Artery of Percheron Infarction: Imaging Patterns and Clinical Spectrum". *American Journal of Neuroradiology* 31.7 (2010): 1283-1289.
5. Raizada K., *et al*. "Non-arteritic anterior ischemic optic neuropathy". NCBI Bookshelf, Stat pearls (2023).
6. Kuybu O., *et al*. "Posterior cerebral artery stroke". NCBI Bookshelf, Stat pearls (2023).
7. Song Y. "Topographic patterns of thalamic infarcts in association with stroke syndromes and aetiologies". *Journal of Neurology, Neurosurgery, and Psychiatry* 82.10 (2011): 1083-1086.

8. Lee LJ., *et al*. "Impact on stroke subtype diagnosis of early diffusion-weighted magnetic resonance imaging and magnetic resonance angiography". *Stroke* 31.5 (2000): 1081-1089.

9. Vogels V., *et al*. "Deep cerebral perforators: Anatomical distribution and clinical symptoms". *Stroke* 52.10 (2021): 660-674.

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